MEDICAL STAFF CONFERENCE

The Pathogenesis, Diagnosis, And Treatment of Pulmonary Embolus

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California, San Francisco. Taken from transcriptions, they are prepared by Drs. Sydney E. Salmon and Robert W. Schrier, Assistant Professors of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine.

DR. SLEISENGER:* The use of anticoagulant drugs in pulmonary embolism is established, but pulmonary embolism continues to be one of the more serious and more important clinical conditions. Dr. Abdallah will present a case which illustrates some of the clinical problems associated with pulmonary embolism.

DR. ABDALLAH:† This was the sixteenth admission to this hospital of a 57-year-old Caucasian woman who had a long history of rheumatic heart disease, hypertension, coronary artery disease, gout and adult-onset diabetes mellitus. The chief complaints on admission were dyspnea and chest pain. Her present illness dated back to her first pregnancy, when she had an episode of thrombophlebitis. She was last in hospital here five months ago with severe congestive heart failure and vague chest pains. At that time diagnosis of pulmonary embolus was made mainly on the basis of changing lung scans. Anticoagulants were administered, and she was discharged after sodium warfarin (Coumadin®), digoxin and diuretic therapy was initiated. However, because of hemorrhagic complications the Coumadin was discontinued three months before the present hospital admission. During the month before admission, the patient had increasing dyspnea, orthopnea, paroxysmal nocturnal dyspnea and a 25-pound gain in weight. Two days before admission she noted pleuritic left anterior chest pain and increasing dyspnea. The past medical history was significant in that in 1956 she had a transient right hemiparesis, and in 1957 a probable acute myocardial infarction. In 1957 she was found to have the murmurs of mitral stenosis and insufficiency and atrial fibrillation. In 1960 closed mitral commisurotomy was carried out. From 1961 to 1969 she had multiple hospital admissions, mainly for episodes of congestive heart failure. In 1969 right and left sided cardiac catheterization were compatible with primary left ventricular failure.

On physical examination the patient was sitting upright and was in obvious respiratory distress. Blood pressure was 170/90 mm of mercury, the pulse was irregular at 60 beats per minute and respirations were 22 per minute. The patient was afebrile. Grade II Keith Wagner retinopathy and jugular venous distension were noted. Examination of the chest revealed a prolonged expiratory phase with diffuse wheezes and scattered rales at the left base. Cardiac examination demonstrated cardiomegaly, murmurs of mitral stenosis and in-

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sufficiency, an opening snap, and a loud pulmonic second sound. The liver was tender and palpable 6 cm below the right costal margin. Peripheral edema and a tender right calf with a positive Homan's sign were also present. The remainder of the examination was within normal limits.

Pertinent laboratory data included a hematocrit of 38 volumes percent, a normal white blood cell count, sodium of 148 mEq, potassium of 2.7 mEq, chloride of 101 mEq and CO_2 of 30 mEq per liter, and creatinine of 1.3 mg per 100 ml. Serial transaminase determinations were within normal limits. An electrocardiogram showed atrial fibrillation, left ventricular hypertrophy, ST-T wave changes of ischemia and U waves. An x-ray film of the chest showed increased cardiomegaly and redistribution of blood flow to the upper lobes. The lung scan performed on the day after admission showed improvement of the left lateral defect present five months earlier. A clinical diagnosis of congestive heart failure and pulmonary embolus was made. Treatment, including heparin and diuretic administration, bed rest and salt restriction, was initiated. The patient showed gradual improvement and was discharged after three weeks.

DR. SLEISENGER: Thank you, Dr. Abdallah. We have asked Dr. Murray, Chief of the Chest Unit at San Francisco General Hospital, to discuss this patient. Dr. Murray has had long experience with this particular disease.

DR. MURRAY:* Thank you Dr. Sleisenger. The subject of pulmonary embolism has been talked about extensively the past few years and is generally introduced with the remarks that it is widely known to be the most commonly missed major medical diagnosis and that it is an extremely common finding in routine autopsy in general hospitals. Pulmonary embolism has also been implicated as the major cause of death, or at least a major contributing cause of death, in such routine autopsies. In the autopsy series the incidence of observed pulmonary embolism certainly was much higher than the incidence of clinically diagnosed pulmonary embolism.[1] I will try to emphasize some of the more unusual manifestations of pulmonary embolism which should alert you to its possible presence, following which appropriate diagnostic procedures can be carried out and treatment begun. The incidence of pulmonary embolism is very difficult to determine. Some authorities have speculated that some embolization is going on all the time, even in perfectly healthy persons. It is believed that aggregates of platelets and fibrin are filtered out as they pass through the lungs, but that these are few in number so that few vessels are blocked and the neurohumoral consequences that accompany the arrival of large clots in the lungs are absent.

Clinically important pulmonary embolism begins with the formation of a thrombus somewhere in the peripheral venous system or in a vascular cavity. After the thrombus is formed numerous large fragments may be shed into the venous system and travel to the lungs. In the middle of the nineteenth century Virchow first identified the three factors that are thought responsible for the development of thrombosis: vascular damage, hypercoagulability of blood, and stasis of blood flow. Injury to the endothelium from trauma or operation is obviously an important contributing cause since the damaged site serves as the nidus for fibrin deposition and the formation of a thrombus. Hypercoagulable states of the blood also deserve mention, although they remain an enigma. They are often hard to detect in the laboratory, but have been documented clinically in disorders such as polycythemia rubra vera in which both the number of red blood cells and platelets are substantially increased. We now also believe that a number of drugs, particularly estrogens either alone or in combination in the birth control pill, can introduce a hypercoagulable state. Stasis very commonly accompanies the development of thrombosis and may result from bed rest, immobilization of an extremity, or from an incompetent venous system in the lower extremities. Stasis is not always associated with embolic disease even though clots form in peripheral vessels, as they can either be lysed *in situ* or can become densely adherent to the vessel wall. If a part breaks off, travels to the lungs and lodges in a branch of the pulmonary artery, the fate of the lung rests on the adequacy of the bronchial collateral circulation.

The bronchial circulation originates from the aorta and nourishes the entire tracheobronchial tree down to the terminal bronchioles. The respiratory bronchioles, alveolar ducts and alveoli normally receive their oxygen and metabolic substrates from the pulmonary arterial circulation. At the junction of the terminal bronchi and respiratory bronchioles there is a rich anastomosis

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among capillaries supplied by both bronchial arteries and the pulmonary arteries. It has been shown that there is a reciprocal relationship between blood flow in the pulmonary arterial circulation and the bronchial circulation, so that if something happens to diminish the perfusing pressure in one avenue of circulation, flow through the other will increase. It follows, therefore, that if there is obstruction to the pulmonary arterial inflow, the bronchial arterial inflow will increase; and the viability of lung tissue subserved by the occluded pulmonary arterial circulation depends thus on the number and patency of the capillary communications.

When we consider pulmonary infarction, which represents ischemia and death of lung tissue, we must consider associated conditions that compromise the ability of the bronchial circulation to increase its blood flow and maintain the metabolic needs of the lung tissue formerly supplied exclusively by the pulmonary arteries. We must anticipate pulmonary infarction in the following three clinical conditions in which the bronchial circulation is often compromised:

1. Congestive heart failure. Presumably because of the increased pressure in the left ventricular or left atrium (into which much of the bronchial venous drainage occurs), there is a lower pressure gradient resulting in reduced blood flow through the bronchial circulation.

2. Chronic pulmonary disease. Changes in available pathways may result from attrition or destruction of bronchial capillaries in chronic pulmonary disease and may thereby prevent an increase in bronchial blood flow after pulmonary arterial occlusion.

3. Hypotension. The patient in shock has a reduced systemic arterial pressure and obviously cannot adequately perfuse blood through the bronchial circulation to maintain the nutritive requirements of an occluded portion of lung.

The incidence of pulmonary infarction following embolism is difficult to determine owing to lack of information about the total number of embolic events. Probably only 5 to 10 percent (or even less) of pulmonary emboli lead to infarction of lung tissue. The diagnosis of an infarct is usually easy since infarction causes characteristic symptoms, including pleuritic chest pain and hemoptysis, and is responsible for the parenchymal infiltrations and pleural effusions seen on x-ray films.

TABLE 1.—*Pulmonary Embolism: X-ray Findings*

Dilatation of Central Pulmonary Arteries
"Cutoff" of Peripheral Pulmonary Arteries
Right Ventricular Dilatation

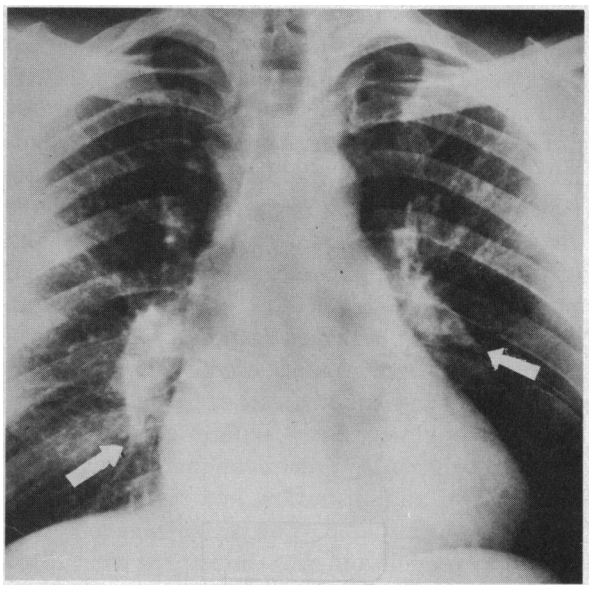

Figure 1.—Chest x-ray of a man with multiple pulmonary emboli showing dilatation of the main pulmonary arteries and right ventricular outflow tract, cutoff of branches to the right and left lower lung fields (arrows), and right ventricular enlargement.

The diagnosis of pulmonary embolus is much more complicated because it does not produce death and ischemia of lung tissue; therefore, hemoptysis, chest pain, pleural effusion and the other classic features of infarction are absent. There are some well-recognized laboratory methods of identifying pulmonary embolism. The two most useful, and most widely advertised, are the electrocardiogram and the chest x-ray film. Table 1 lists the most valuable criteria for the diagnosis of pulmonary embolism by conventional chest x-ray studies, and Figure 1 shows an example of an advanced case. When the x-ray pattern of abrupt pulmonary artery termination and vascular dilatation is present, one should have no difficulty in making the diagnosis of multiple pulmonary emboli. When any of the characteristic x-ray findings are present, one should be very suspicious about pulmonary embolism. Unfortunately, x-ray films of the chest are often normal and of very little value in the diagnosis of the vast majority of the patients who have pulmonary embo-

TABLE 2.—*Pulmonary Embolism: Electrocardiogram Findings*

S_1 Q_3 and $S_{1,2,3}$
Inverted T Waves V_{1-3}
Clockwise Rotation
Right Bundle Branch Block

TABLE 3.—*Pulmonary Embolism: Clinical Features*

Central Nervous System: Anxiety, Restlessness, Syncope
Cardiac: Congestive Failure, Shock, Arrhythmias
Pulmonary: Hypoxia, Edema, Wheezing
General: Dyspnea, Fever

lism. If the changes are evident, fine; if they are not, one certainly cannot exclude the diagnosis.

The same considerations hold for the electrocardiogram. Table 2 presents the classic electrocardiogram criteria for acute pulmonary embolism, and Figure 2 shows a tracing from a patient with a classical pattern. If either this pattern or one of the other diagnostic EKG patterns is present, one should have no trouble in making the diagnosis of acute cor pulmonale—the most common cause of which is pulmonary embolism. I wish to emphasize again that, like the x-ray film, the EKG is very often nonspecific. It may, in fact, be normal in the presence of severe massive pulmonary embolism. Therefore, one cannot rely on it as a good technique for establishing the diagnosis.

Then how do we make the diagnosis of pulmonary embolism? Much has to be done on the basis of clinical suspicion. Table 3 lists the major manifestations of pulmonary embolism that can be related to involvement of three organ systems: the brain, the heart and the lungs. The central nervous manifestations may be prominent—in fact, sufficiently predominant to mislead the attending physician. Anxiety and restlessness are extraordinarily common in patients who have pulmonary embolism; syncope is related to the transient fall in arterial pressure, the mechanisms for which is discussed below. If syncope is prolonged, it can be associated with seizures; and occasionally, if the hypotension persists, focal areas of cerebral ischemia can develop so that the patient appears to have had a stroke.

The heart is involved both directly and indirectly. I have already mentioned acute cor pulmonale resulting from the sudden alteration in pulmonary arterial pressure. There are two mechanisms that act in concert to generate the increase in pulmonary arterial pressure. One of these is

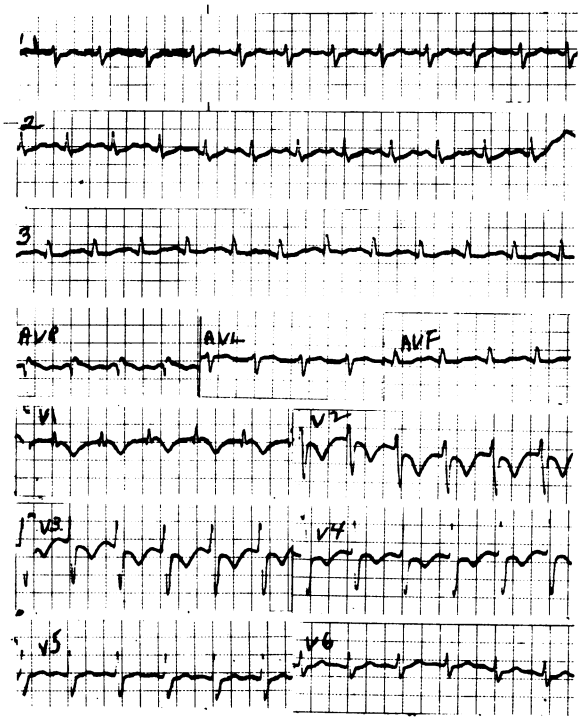

Figure 2.—Electrocardiogram of a patient with an acute pulmonary embolus showing S_1 Q_3, inverted T waves V_{1-3}, and clockwise rotation.

the mechanical presence of the embolus in the pulmonary vasculature, so that there is an obstruction to part of the vascular pathways available to the blood flow. Since a pulmonary artery can be ligated in a normal individual without causing much, if any, elevation in pulmonary arterial pressure, it is generally believed that from 60 to 70 percent of the total cross-sectional area of the pulmonary vascular bed has to be obstructed before the pulmonary arterial pressure increases. Since the number and size of the pulmonary arteries involved (that is, the reduction in cross-sectional area of the vascular bed from occlusion) is often insufficient to account for a rise in pulmonary arterial pressure, other factors affecting blood vessels must be considered. Attention has been directed toward the possibility of pulmonary arterial vasospasm from either reflex or humoral stimuli.

Shock also seems to depend upon two mechanisms that act in concert and contribute to the fall in arterial pressures. One of these mechanisms is reduced cardiac output, which can be related in part to obstruction of the pulmonary vascular bed. In other conditions, a fall in cardiac output of the magnitude usually observed in pulmonary embolus is adequately compensated by peripheral

vasomotor reflexes which maintain arterial pressure. As these reflexes appear absent in pulmonary embolus, we infer that there is a second mechanism, mediated through reflexes, that causes systemic vasodilation. The fall in cardiac output, plus the vasodilatation, leads to the rather striking reduction in systemic arterial pressure. The fall in blood pressure leads to a reduction in cerebral perfusion and is undoubtedly the mechanism leading to the central nervous system abnormalities. Additionally, paroxysmal arrhythmias (usually superventricular) can also accompany the lodging of a clot in the pulmonary vasculature.

A number of abnormalities related to pulmonary function also deserve comment. The most common of these is hypoxia, but its mechanism is still under some dispute; certainly it depends upon the development of a more generalized pulmonary abnormality than occurs in the lung subserved by the occluded vessel. In one study of patients with embolism, hypoxia resulted from a combination of ventilation-perfusion abnormalities and right-to-left shunting of blood.[2] Pulmonary edema has been reported in association with pulmonary emboli and may be due to arrhythmias or tachycardias induced in patients who have underlying heart disease. Interestingly, pulmonary edema has also been observed in patients without underlying heart disease, indicating that other mechanisms (to be discussed below) must account for more generalized changes in hydrostatic forces or vascular permeability.

Wheezing is also an occasional accompaniment of recurrent or single large pulmonary emboli.[3] The mechanisms for all of these—hypoxia, pulmonary edema, and wheezing—cannot be accounted for simply on the basis of a local phenomenon related to the clot in a branch of the pulmonary arterial system. The generalized nature of these responses implies that something else occurs which makes the remainder of the lung responsive to the embolic event. A number of factors have been proposed in an attempt to explain the generalized response. Dr. Jay Nadel[4] and coworkers of the Cardiovascular Research Institute here have demonstrated that histamine is released following emboli to the lungs. Dr. Julius Comroe[5] and others[6] believe that serotonin may be involved in some of the subsequent vascular and bronchial events that take place. Fibrinopeptides and other vasoactive substances are probably also released in the embolic process. Whether these

TABLE 4.—*Pulmonary Embolism: "Unusual Manifestations"*

Unexplained Hyperventilation or Dyspnea
Unexplained Fever
Fever Not Responding to Antibiotics
Increase in Severity of Congestive Heart Failure
Tachycardia, Digitalis Toxicity and Refractory Heart Failure
Unexplained Leukocytosis

TABLE 5.—*Pulmonary Embolism: Aids in Diagnosis*

Hints
Clinical Features
Electrocardiogram Changes
X-ray Changes
Moderately Useful
Perfusion Lung Scans
Pulmonary Function Studies
Diagnostic
Pulmonary Angiography

circulate through the vascular system and back to the lung, where they cause the generalized phenomena that I mentioned, or initiate various reflex mechanisms is uncertain at this stage. Neurohumoral processes of some sort must account for the diffuse pulmonary phenomena following a focal embolus.

Two of the most common clinical features of pulmonary embolus are dyspnea and fever (Table 3). Dyspnea is the most common symptom, and it could originate from abnormalities in the central nervous system, the cardiovascular system or the respiratory system. Regardless of its origin, dyspnea, particularly that which is recurrent or episodic, should call attention to the possibility of underlying pulmonary embolism. The most common sign is fever, which is usually low grade; but at times it may be 39.4° C (103.0° F) or higher and may have a hectic course that resembles the fever of sepsis. In a large series of cases with pulmonary embolism, fever was present in approximately 80 percent.[1]

The so-called "unusual manifestations" of pulmonary embolism[7] are listed in Table 4. Some of these features certainly relate to the patient discussed today. If a patient has a clinical condition favoring the formation of venous thrombosis (vascular injury, stasis, increased coagulability of blood) and then any of these clinical manifestations develops, the possibility of pulmonary embolism should be considered.

When pulmonary embolism is suspected, attempts should be made to establish the diagnosis. The relative value of the sources of diagnostic in-

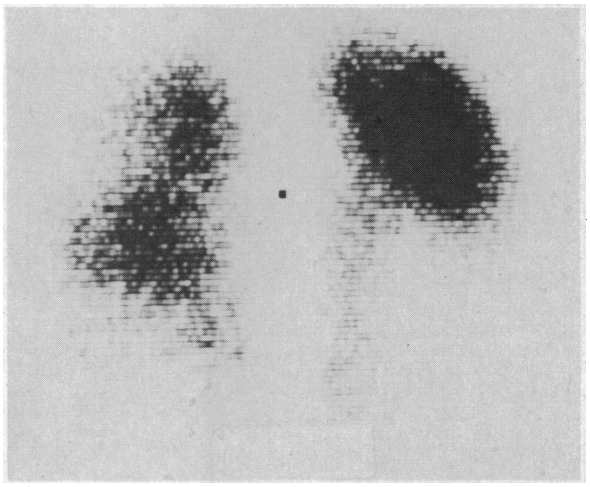

Figure 3.—Perfusion lung scan with macro-aggregated albumin of the same patient whose x-ray is shown in Figure 1. Note the marked diminution of blood flow, shown by the absence of black dots, over the entire right lung and left lower lung field.

formation are given in Table 5. As I mentioned above, hints to the correct diagnosis are offered by clinical features, changes or chest x-ray findings. Additional more confirmatory, but still nonspecific, evidence can be obtained by radioisotope lung scanning (Figure 3) and pulmonary function studies. The only definitive study, however, is pulmonary angiography.

It is tempting to believe that all filling defects seen on perfusion lung scans result from pulmonary arterial occlusive lesions, but such is definitely not the case. Disturbances in blood flow reflected by defects in perfusion lung scans are frequently encountered in patients with pneumonia and pleural effusion; the sites of involvement are usually those where infiltrations or fluid can be identified on the plain chest x-ray film. Vascular filling defects may also be seen when the plain film appears normal in patients with emphysema, bronchitis or asthma. In emphysema there is actually destruction of lung parenchyma and pulmonary capillaries to account for the vascular defects; however, in bronchitis and asthma the blood vessels are intact, but blood flow is redistributed away from regions of the lung where bronchospasm, secretions or edema impair ventilation. The latter conditions may be variable and evanescent, so even a changing lung scan is not pathognomonic of pulmonary emboli. Despite the nonspecificity of positive lung scans, a negative result is strong evidence against significant pulmonary embolism.[8]

In theory, pulmonary function studies should be of great help in the diagnosis of acute pulmonary embolism. We would suspect that since the lung supplied by an occluded blood vessel could not participate in oxygen uptake or carbon dioxide elimination, it would add what we call "wasted ventilation" or dead space. Simple tests could measure the extra amount of wasted ventilation or the difference in carbon dioxide tension between the patient's expired air at the end of expiration and in his arterial blood. (Normally there is no significant difference; and any increase at rest must be due to added dead space.) Although this analysis is theoretically attractive, the lung has a great capacity to redistribute blood flow and ventilation in response to reductions in either blood flow or ventilation to maintain "ideal" gas exchange. Therefore, when there is obstruction to blood flow, secondary changes in the bronchial walls, the smooth muscle of alveolar ducts, and the surface lining of the alveoli, all serve to prevent ventilation of the lung so that the carbon dioxide tension differences and increase in wasted ventilation may not be apparent. Pulmonary function studies in the resting patient are of little value because of the compensatory ability of the lung to redistribute ventilation when blood flow is impaired.

Dr. Nadel et al[9] have studied a large group of patients with pulmonary vascular occlusive diseases and have showed that the compensatory responses can be overcome by exercising the patient. Ordinarily when a subject exercises, his absolute value of wasted ventilation increases slightly, his tidal volume increases substantially, and his ratio of wasted ventilation to tidal volume decreases. When patients with pulmonary vascular diseases take deep breaths during exercise, they begin to ventilate the areas that were poorly ventilated at rest. The reason is that deeper breathing maneuver increases transpulmonary pressure and "opens" airways and alveoli that were "closed" when breathing at normal tidal volumes. Exercise testing is an extremely useful maneuver, and is one of the most sensitive tests available for the diagnosis of chronic pulmonary vascular occlusion. However, it is of little value in the clinical situations we are discussing because most of the patients in whom one is suspicious of pulmonary embolism are not suitable subjects for exercise studies.

The only way the definitive diagnosis of pulmo-

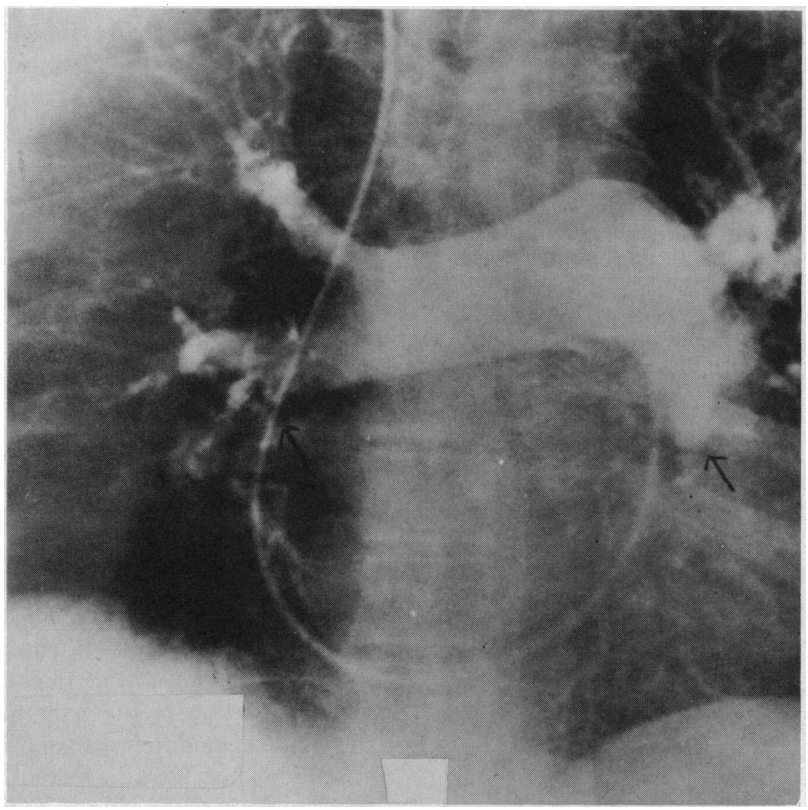

Figure 4.—Pulmonary angiogram of the same patient whose chest x-ray and lung scan are shown in Figures 1 and 3. Note the marked reduction in pulmonary arterial blood flow to the entire right lung and left lower lobe.

nary embolism can be made is by pulmonary angiography. (See Figure 4.) Routine angiography is only adequate for the detection of emboli in vessels down to 3 or 4 mm in diameter. Dr. Richard Greenspan, of the Department of Radiology at this hospital, has developed the use of small focal spots and magnification techniques that allow us to visualize blood vessels down to approximately 1 mm in diameter; and this adds a very important refinement to our ability to examine the sites and magnitude of pulmonary vascular occlusive disease. It is very important to do pulmonary angiography within the first 12 to 24 hours in the clinical course of a patient with suspected pulmonary emboli. Serial angiographic studies have shown that pulmonary emboli will lyse or fragment and move into smaller peripheral vessels very quickly; therefore, a negative angiogram two to three days after a suspected event does not tell you whether or not the patient had an earlier episode.

This entire discussion has emphasized early and accurate diagnosis so that treatment can be initiated. Available modalities of treatment are listed in Table 6. Many are based on the premise that if you can keep emboli from recurring, the lung has a relatively good capacity—in fact, a surprisingly good capacity—to cope with the emboli that have already arrived there. The lung handles clots by fibrinolytic mechanisms that cause dissolution and fragmentation of clots and by recanalization of the vessels. Vessels that have been completely occluded can have blood flow restored and perform quite normally. Until recently the whole emphasis on treatment has been on prevention of further embolization by anticoagulants and surgical procedures. Now we have a new agent, urokinase, that may accelerate and augment the dissolution of emboli in the lung and the thrombus in the peripheral vessel. A cooperative study is evaluating the results of trials of urokinase, and although preliminary results are encouraging, the final results are not yet available.

TABLE 6.—*Pulmonary Embolism: Treatment*

Anticoagulants
Heparin
Vena Cava Ligation
Urokinase
Surgical Removal

DR. SLEISENGER: Thank you, Dr. Murray, for that complete and concise summary of pulmonary embolism and infarction.

A PHYSICIAN: Is occlusion of the bronchial arterial circulation responsible for infarction?

DR. MURRAY: It is very difficult to assess accurately the magnitude of bronchial blood flow and its distribution. Its role in pulmonary infarction is largely inferential. Pulmonary infarcts develop in clinical circumstances in which there is suggestive evidence of an impairment of bronchial circulation; but solid documentation has not been obtained. The normal role of the bronchial circulation remains obscure, as evidenced by lung transplantation studies. Removal of a lung from an animal and reimplantation completely interrupts all of the bronchial blood flow, and yet nothing adverse appears to happen to the airways. Perhaps their metabolism is maintained by retrograde flow from the pulmonary arterial circulation, but no one yet knows why the lung survives.

A RADIOLOGIST: Recent observations by Dr. John Austin and Dr. Stuart Sagel in the Department of Radiology here may be relevant to the wheezing described in the patient presented. In their studies of experimental pulmonary embolism, they used a small focal spot tube and direct 10-fold magnification after tantalum bronchography. They have shown rather striking and dramatic constriction of bronchi all the way down to the very small bronchial level, both ipsolateral and contralateral to unilateral pulmonary emboli.

DR. MURRAY: That is an interesting observation that could relate to the ventilation-perfusion abnormalities which lead to hypoxia as well as to wheezing resulting from bronchoconstriction.

TRADE AND GENERIC NAMES OF DRUGS

Coumadin® ..... sodium warfarin

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TRACHEOTOMY HAZARDS IN TRAUMA TO THE TRACHEAL-LARYNGEAL REGION

"Car accident victims can sometimes have a tremendous amount of damage to the laryngeal-tracheal complex with very little superficial evidence, that is, in the soft tissues of the neck. The point is that tracheotomy is not the real solution. If a tracheotomy is necessary, then endeavors should be made immediately to find out why. . . . So often in these cases, the tracheotomy is performed by someone who is not particularly knowledgeable about this type of injury, and that is the end of it until he tries to extubate the patient. Then he finds that he's in trouble because of obstruction. . . .

"Another reminder: when patients with acute laryngeal trauma come to the emergency room and tracheotomy is necessary, one must splint the neck; movement must be minimized."

—G. SLAUGHTER FITZ-HUGH, M.D., Charlottesville, Va.

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